

**Applicant** : Neil H. Bander                                      **Art Unit** : 1642  
**Serial No.** : 09/357,704                                        **Examiner** : Gary Nickol  
**Filed** : July 20, 1999  
**Title** : TREATMENT AND DIAGNOSIS OF PROSTATE CANCER

DECLARATION UNDER 37 CFR 1.131

1. I am the inventor of the subject matter disclosed and claimed in the above-referenced United States Patent Application.
2. I am familiar with the present claims of the application, which are directed to a method of treating, preventing, or delaying development or progression of prostate cancer in a subject.
3. Prior to March 25, 1996, I had conceived my invention as described and claimed in the above-identified application in this country, a NAFTA country or WTO country, and had diligently reduced the invention to practice, as evidenced below.
4. I submit herewith Exhibits A-L, evidence showing conception of the claimed invention prior to the March 25, 1996.

Exhibit A shows an excerpt from a document describing my research on antibodies and their use in cancer that I wrote prior to March 25, 1996. The document dates, dates within the text of the document, and the name of the individual to whom the document is addressed, have been redacted in the excerpt provided in Exhibit A. The document clearly demonstrates that I was actively pursuing monoclonal antibodies for clinical use in prostate cancer.

As indicated in that document, my laboratory had been testing several monoclonal antibodies for their ability to bind to live LNCaP cells, a human prostate cancer model cell line. Several of the antibodies I had already characterized had demonstrated excellent potential, in that they were able to lyse LNCaP cells *in vitro* in the presence of human serum as a source of complement.

I had demonstrated that the antibodies localized to prostate cancer and sites of metastatic disease (in the lymph nodes and liver). More importantly, I had demonstrated that administration of these antibodies to human subjects resulted in treatment and prevention of prostate cancer. For example, I demonstrated that decreased PSA levels were seen upon administration of these antibodies to human subjects, and that there were no signs of relapse seen in these subjects. All of this is shown in Exhibit A. Exhibit A does not discuss the specific antibodies of the invention. In connection with the work described in Exhibit A, I characterized other monoclonal antibodies including the antibodies of the invention. This is shown in Exhibits B-L below.

Exhibit B, is a tag from a mouse cage in my (Dr. Bander's) laboratory indicating that mice immunized with LNCaP cells as part of "Fusion E" experiments were received, immunized, and given a final booster. Dates on the tag have been redacted. The dates show the work was done prior to March 25, 1996.

Exhibits C-L discussed below all show pages from notebooks from my (Dr. Bander's) laboratory. The date on each of the pages is redacted. Each page is dated prior to March 25, 1996.

Exhibit C is an entry from a laboratory notebook in my (Dr. Bander's) laboratory. This entry shows rosette and cytotoxicity studies of the fusion E antibodies including monoclonal antibody E99 (which is an antibody of the invention), and demonstrates that monoclonal antibody E99 binds to LNCaP cells, a human prostate cancer cell line.

Exhibit D is an entry from a laboratory notebook in my (Dr. Bander's) laboratory. This entry demonstrates that E99 binds renal tubules very weakly, and binds prostate cancer cells strongly.

Exhibit E is an entry from a laboratory notebook in my (Dr. Bander's) laboratory. This entry demonstrates that E99 binds to benign hyperplastic prostate tissue obtained from various human patients. E99 was detected using a fluorescein label.

Exhibit F is an entry from a laboratory notebook in my (Dr. Bander's) laboratory. This entry demonstrates that E99 is an IgG<sub>3</sub> class antibody.

Exhibit G is an entry from a laboratory notebook in my (Dr. Bander's) laboratory. This entry demonstrates that the E99 antibody is capable of lysing PSMA-expressing LNCaP cells, but not PSMA-negative PC3 and Du145 cells.

Exhibit H is an entry from a laboratory notebook in my (Dr. Bander's) laboratory. This entry demonstrates that E99 binds strongly to prostate cancer and benign hyperplastic prostate tissues from human patients, binds normal kidney proximal tubules weakly, and does not bind at all to normal liver, lung, pancreas, testis, esophagus, uterus, small bowel, stomach, thyroid, or spleen.

Exhibit I is an entry from a laboratory notebook in my (Dr. Bander's) laboratory. This entry demonstrates that E99, J415 and J533 (all of which are antibodies of the invention) bind kidney proximal tubules and LNCaP prostate cancer cells, a human prostate cancer cell line, but do not bind normal colon.

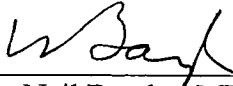
Exhibit J is an entry from a laboratory notebook in my (Dr. Bander's) laboratory. This entry demonstrates that E99 and J591 (both of which are antibodies of the invention) bind kidney proximal tubules and LNCaP prostate cancer cells, but do not bind normal colon.

Exhibit K is an entry from a laboratory notebook in my (Dr. Bander's) laboratory. This entry demonstrates that the J415, J533, and E99 antibodies bind weakly to proximal tubule cells of the normal kidney, and bind benign hyperplastic prostate and cancerous prostate tissue from human patients, and that the J415 and J533 antibodies do not bind at all to normal liver, small intestine, or lung.

Exhibit L is an entry from a laboratory notebook in my (Dr. Bander's) laboratory. This entry demonstrates monoclonal antibodies including E99, J415, J533 and J591 bind to prostate cancer tissue from a number of individuals.

5. Exhibit A demonstrates that I had conceived of using monoclonal antibodies in the treatment and prevention of prostate cancer and Exhibits B-L demonstrate that antibodies which bind PSMA and recognize benign hyperplastic, and cancerous prostate cells from human patients were produced for clinical use in human subjects prior to March 25, 1996. In sum, I submit evidence herewith that shows conception of the claimed invention prior to March 25, 1996.
6. Very shortly after March 25, 1996, namely, May 6, 1996, just a little more than 5 weeks later, the claimed methods were constructively reduced to practice upon filing of provisional application 60/016,976 from which the above-identified application claims priority.
7. I hereby declare that all statements made herein of my own knowledge are true and that all statements made on information and belief are believed to be true; and further that these statements were made with the knowledge that willful false statements and the like are punishable by fine or imprisonment, under Title 18 § 1001 of the United States Code, and that such willful false statements may jeopardize the validity of the application or any patent issuing thereon.

10/7/02  
Date

  
Neil Bander, MD

[REDACTED]

Particularly when viewed in the context of the advances in the mAb therapy field in general, the attributes and strengths of mAbs are particularly well-suited to the demands of prostate cancer therapy:

1. mAbs can specifically localize to disseminated tumor sites at levels orders of magnitude higher than normal tissues.
  2. Therapeutic efficacy has been proven in tumor types (e.g., colon cancer and NHL) where the clinical setting resembles prostate cancer.
  3. mAbs have a number of potential mechanisms of anti-tumor activity including:
    - a. the relative radiosensitivity of PCa provides one potential class of cytotoxic agents to specifically deliver to tumor sites by way of mAb.
    - b. mAbs can trigger the host's own immune response to tumor.
  4. Prostate cancer metastases are small-volume sites (typically measured in microns or mm) ideal for radioisotope or immunotherapy.
  5. The availability of established parameters such as PSA and pathological features (e.g., stage, Gleason score, seminal vesicle invasion, positive margins, nodal disease, etc.), provide appropriate indications for adjuvant mAb therapy where such therapy is likely to be most beneficial.
  6. Last, but not least, is the fact that mAbs are non-toxic.
- [REDACTED]
- [REDACTED]

We believe that we are well on the way to prove that these advantages are more than just theoretical. We have recently completed our mAb Prost 30 biodistribution study in 15 patients with prostate cancer. Doses ranged from 1.0 to 20.0 mg of mAb. Fourteen of the 15 patients had their prostates *in situ* and were evaluable for localization of Prost 30. In all 14 of these cases, including two with prior radiation therapy, the prostate was successfully imaged. In two cases, patients had known sites of metastatic disease imaged on conventional CT scans: regional lymph nodes (both patients) and liver (1 patient). In these cases, these sites also were successfully imaged with Prost 30. In four cases, after resecting the prostate one week after mAb administration, the prostate specimens were scanned alongside specimens of blood drawn at the time of the resection (see appended representative figure). These studies confirmed specific uptake in the prostate at substantially higher levels than in the blood, and this uptake persists for more than one week. No patient on the trial had any side effects.

unconjugated ("naked") Prost 30 in a series of patients evaluable for response. This was to establish the safety of naked antibody-as a prelude to an adjuvant trial similar to that already shown effective in colon cancer -- and to provide a benchmark with which to compare the results of a radiolabeled mAb trial. Doses range from 1.25 mg to 5.0 mg -- the level at which we saw the responses in the earlier trial. Fifteen weeks into this trial we have entered 16 patients. Many of the patients, including some at the lowest dose level, have responded with declines in PSA ranging from 25-55%. It is obviously too early to discuss duration of response. While this data is exceedingly preliminary, it is certainly provocative, particularly as the mAb has no conjugated cytotoxic moiety.

We have also developed in the laboratory a higher affinity Prost 30 mAb which we have designated Prost 130. Prost 130 binds the same antigen as Prost 30, but at a different, and repeated, site. It is possible that these mAbs (Prost 30 and 130) will be additive or synergistic in combination. Two other mAbs we have recently developed, C37 and C219, have demonstrated both prostate specificity (in vitro and in vivo) and the ability to directly lyse LNCaP cells in vitro in the presence of human serum as a source of complement. Furthermore, the cytotoxicity of these mAbs are synergistic when combined in vitro.

synergistic when combined in vitro. [REDACTED] these three mAbs (Prost 130, C37 and C219) have been contracted to an FDA-approved manufacturer for production of clinical grade material for upcoming trials.

hibit B

FORM BY MEN LAB & LABEL BOX AT ALBERTSON NY 11507  
1183

Dr  
Bander



Fusion, E

COST  
CENTER # 6801

CAGE ID # 92080 RM. #         

INVESTIGATOR                      EXT. 65499

SPSTR. BALB/c SEX ♀

DATE RECD.              PO #         

ID # OR  
#CAGE                      VENDOR                     

Dr Lin Peggy

LNcap cell 6x10<sup>6</sup>/per mouse i.p

date             

Fusion: date              i.p

date              i.p

ANIMAL MAINTENANCE  
CHARGES TO C.C.                     

FUND#                     

PROTOCOL#                     

final breeder:

Cytotoxicity for Lncap

plate 1

91	92	93	94	95	96	97	98	99	100
-	-	-	-	-	-	-	-	-	-
101	102	103	104	105	106	107	108	109	110
-	-	-	-	-	-	-	-	-	-
111	112	113	114	115	116	117	118	119	120
-	-	-	-	-	-	-	-	-	-
121	122	123	124	125	126	127	128	129	130
-	-	-	-	-	-	-	-	-	-
131	132	133	134	135	136	137	138	139	140
-	-	-	-	-	-	-	-	-	-
C37									Rem

Fusion E

plate 2

141	142	143	144	145	146	147	148	149	150
-	-	-	-	-	-	-	-	-	-
151	152	153	154	155	156	157	158	159	160
-	-	-	-	-	-	-	-	-	-
161	162	163	164	165	166	-	-	-	-
-	-	-	-	-	-	-	-	-	-
C37									Rem

Fusion E

Plate 4

141	142	143	144	145	146	147	148	149	150
-	-	-	-	-	-	-	-	-	-
151	152	153	154	155	156	157	158	159	160
-	-	-	-	-	-	-	-	-	-
161	162	163	164	165	166	-	-	-	-
-	-	-	-	-	-	-	-	-	-
w/									653

Reset test for Lncap  
plate 3

91	92	93	94	95	96	97	98	99	100
-	-	-	-	-	-	-	-	-	-
101	102	103	104	105	106	107	108	109	110
-	-	-	-	-	-	-	-	-	-
111	112	113	114	115	116	117	118	119	120
-	-	-	-	-	-	-	-	-	-
121	122	123	124	125	126	127	128	129	130
-	-	-	-	-	-	-	-	-	-
131	132	133	134	135	136	137	138	139	140
-	-	-	-	-	-	-	-	-	-
w/									653

Terasaki Plate

Exhibit C

E. 21617 D

Side	Section	Morb	Result		
1	NK, N colon, Pca (Kortey)	E 91	+	+	+
2		E 93	+	+	+
3		E 94	+	+	+
✓ 4		E 99	tube (+)	-	+
5		E 100	-	-	-
6		E 101	+	+	+
7		E 102	-	-	-
8		E 105	+	+	+
9		E 109	+	+	+
10		E 116	-	+	+
★ V 11		E 119	-	-	+
12		E 120	+	+	+
13		E 121	+	-	+
14		E 130	+	+	+
15		E 131	+	+	+
16		E 132	+	+	+
17		E 133	+	+	+
18		E 134	+	+	+
19		E 135	-	-	-
20		E 136	+	+	+
21		E 141	+	+	+
22		E 144	+	+	+
23		E 147	+	+	+
24		E 148	-	-	-
25		E 152	+	+	+
26		E 154	+	+	+
27		E 163	-	-	-
16 28		Fil 1/2 P410	+	+	+



# Exhibit E

Slide	Section	Micro		Result	
		ascite	ascite	epith	stroma
1	BPH (Friedlander)	C37 1:100	C219 1:100	+	+
2		E27	E99		
3		653	P410	—	+
4		C37 ascite 1:100	C219 ascite 1:100	—	—
5	FIT-RAM	E27	E99	—	—
6		653	P410	—	+
7	BPH (Rodriguez, J)	C37 ascite 1:100	C219 ascite 1:100	epith +	stroma +
8		E27	E99	+	+
9		653	P410	—	+
10		C37 ascite 1:100	C219 ascite 1:100	—	+
11	FIT-RAM	E27	E99	+	weak +
12		653	P410	—	+
13	BPH (CHAN, M. Y)	C37 ascite 1:100	C219 ascite 1:100	epith +	stroma +
14		E27	E99	—	+
15		653	P410	—	+
16		C37 ascite 1:100	C219 ascite 1:100	—	—
17	FIT-RAM	E27	E99	—	—
18		653	P410	—	+
19	BPH (Deere, J)	C37 ascite 1:100	C219 ascite 1:100	epith +	stroma +
20		E27	E99	+	+
21		653	P410	—	+
22		C37 ascite 1:100	C219 ascite 1:100	—	+
23	FIT-RAM	E27	E99	+	+
24		653	P410	—	+

Exhibit F

Plate 1

Eq9

12.7

Eng

١٥٦

[illegible]

E 202

5

653

F 458

$\frac{1}{2}a_1$	$\frac{1}{2}a_2$
$\frac{1}{2}a_3$	$\frac{1}{2}a_4$
$\frac{1}{2}a_5$	$\frac{1}{2}a_6$
$\frac{1}{2}a_7$	$\frac{1}{2}a_8$
$\frac{1}{2}a_9$	$\frac{1}{2}a_{10}$
$\frac{1}{2}a_{11}$	$\frac{1}{2}a_{12}$
$\frac{1}{2}a_{13}$	$\frac{1}{2}a_{14}$
$\frac{1}{2}a_{15}$	$\frac{1}{2}a_{16}$
$\frac{1}{2}a_{17}$	$\frac{1}{2}a_{18}$
$\frac{1}{2}a_{19}$	$\frac{1}{2}a_{20}$
$\frac{1}{2}a_{21}$	$\frac{1}{2}a_{22}$
$\frac{1}{2}a_{23}$	$\frac{1}{2}a_{24}$
$\frac{1}{2}a_{25}$	$\frac{1}{2}a_{26}$
$\frac{1}{2}a_{27}$	$\frac{1}{2}a_{28}$
$\frac{1}{2}a_{29}$	$\frac{1}{2}a_{30}$
$\frac{1}{2}a_{31}$	$\frac{1}{2}a_{32}$
$\frac{1}{2}a_{33}$	$\frac{1}{2}a_{34}$
$\frac{1}{2}a_{35}$	$\frac{1}{2}a_{36}$
$\frac{1}{2}a_{37}$	$\frac{1}{2}a_{38}$
$\frac{1}{2}a_{39}$	$\frac{1}{2}a_{40}$
$\frac{1}{2}a_{41}$	$\frac{1}{2}a_{42}$
$\frac{1}{2}a_{43}$	$\frac{1}{2}a_{44}$
$\frac{1}{2}a_{45}$	$\frac{1}{2}a_{46}$
$\frac{1}{2}a_{47}$	$\frac{1}{2}a_{48}$
$\frac{1}{2}a_{49}$	$\frac{1}{2}a_{50}$
$\frac{1}{2}a_{51}$	$\frac{1}{2}a_{52}$
$\frac{1}{2}a_{53}$	$\frac{1}{2}a_{54}$
$\frac{1}{2}a_{55}$	$\frac{1}{2}a_{56}$
$\frac{1}{2}a_{57}$	$\frac{1}{2}a_{58}$
$\frac{1}{2}a_{59}$	$\frac{1}{2}a_{60}$
$\frac{1}{2}a_{61}$	$\frac{1}{2}a_{62}$
$\frac{1}{2}a_{63}$	$\frac{1}{2}a_{64}$
$\frac{1}{2}a_{65}$	$\frac{1}{2}a_{66}$
$\frac{1}{2}a_{67}$	$\frac{1}{2}a_{68}$
$\frac{1}{2}a_{69}$	$\frac{1}{2}a_{70}$
$\frac{1}{2}a_{71}$	$\frac{1}{2}a_{72}$
$\frac{1}{2}a_{73}$	$\frac{1}{2}a_{74}$
$\frac{1}{2}a_{75}$	$\frac{1}{2}a_{76}$
$\frac{1}{2}a_{77}$	$\frac{1}{2}a_{78}$
$\frac{1}{2}a_{79}$	$\frac{1}{2}a_{80}$
$\frac{1}{2}a_{81}$	$\frac{1}{2}a_{82}$
$\frac{1}{2}a_{83}$	$\frac{1}{2}a_{84}$
$\frac{1}{2}a_{85}$	$\frac{1}{2}a_{86}$
$\frac{1}{2}a_{87}$	$\frac{1}{2}a_{88}$
$\frac{1}{2}a_{89}$	$\frac{1}{2}a_{90}$
$\frac{1}{2}a_{91}$	$\frac{1}{2}a_{92}$
$\frac{1}{2}a_{93}$	$\frac{1}{2}a_{94}$
$\frac{1}{2}a_{95}$	$\frac{1}{2}a_{96}$
$\frac{1}{2}a_{97}$	$\frac{1}{2}a_{98}$
$\frac{1}{2}a_{99}$	$\frac{1}{2}a_{100}$

plate 2

1572 F

1219

**Terraced Plate**

[illegible][illegible]

# Exhibit F (cont)

plate 1

15 min

	1	2	3	4	5	6	7	8	9	10	
	$\frac{1}{2}G_1$	$\frac{1}{2}G_{2a}$	$\frac{1}{2}G_{2b}$	$\frac{1}{2}G_3$	$\frac{1}{2}M$	$\frac{1}{2}G_1$	$\frac{1}{2}G_{2a}$	$\frac{1}{2}G_{2b}$	$\frac{1}{2}G_3$	$\frac{1}{2}M$	plate 1
E27 { F	+0.22	+0.21	+0.18	+0.34	+1.41	+0.23	+0.21	+0.17	+0.92	+0.58	} E99 F E D C B A
E119 { E	+0.23	+0.22	+0.17	+0.30	+1.37	+0.26	+0.22	+0.18	+1.11	+0.51	
E119 { D	+0.94	+0.19	+0.19	+0.35	+1.39	+1.00	+0.71	+0.31	+0.31	+1.41	
E119 { C	+0.85	+0.21	+0.20	+0.32	+1.41	+0.95	+0.23	+0.30	+0.40	+1.47	
E119 { B	+0.21	+0.17	+0.18	+0.23	+0.26	+0.09	+0.10	+0.10	+0.15	+0.11	
E119 { A	+0.18	+0.17	+0.13	+0.26	+0.30	+0.09	+0.10	+0.10	+0.13	+0.10	

plate 2

15 min

	1	2	3	4	5	6	7	8	9	10	
	$\frac{1}{2}G_1$	$\frac{1}{2}G_{2a}$	$\frac{1}{2}G_{2b}$	$\frac{1}{2}G_3$	$\frac{1}{2}M$	$\frac{1}{2}G_1$	$\frac{1}{2}G_{2a}$	$\frac{1}{2}G_{2b}$	$\frac{1}{2}G_3$	$\frac{1}{2}M$	plate 2
E455 { F	+0.30	+0.26	+0.22	+0.36	+1.43	+0.38	+0.26	+0.22	+0.39	+1.41	} E102 F E D C B A
E455 { E	+0.24	+0.23	+0.21	+0.48	+1.43	+0.43	+0.25	+0.17	+0.35	+1.40	
E37 { D	+0.29	+0.27	+0.20	+0.34	+1.43	+0.30	+0.28	+0.20	+0.32	+1.42	
E37 { C	+0.28	+0.25	+0.19	+0.34	+1.43	+0.25	+0.23	+0.20	+0.32	+1.40	
E37 { B	+0.26	+0.19	+0.14	+0.26	+0.32	+0.10	+0.11	+0.09	+0.12	+0.13	
E37 { A	+0.24	+0.21	+0.13	+0.27	+0.31	+0.09	+0.12	+0.09	+0.29	+0.09	

plate 1

60 min

	1	2	3	4	5	6	7	8	9	10	
	$\frac{1}{2}G_1$	$\frac{1}{2}G_{2a}$	$\frac{1}{2}G_{2b}$	$\frac{1}{2}G_3$	$\frac{1}{2}M$	$\frac{1}{2}G_1$	$\frac{1}{2}G_{2a}$	$\frac{1}{2}G_{2b}$	$\frac{1}{2}G_3$	$\frac{1}{2}M$	plate 1
E27 { F	+0.48	+0.46	+0.36	+0.81	+1.41	+0.50	+0.46	+0.28	+1.38	+1.19	} E99 F E D C B A
E27 { E	+0.52	+0.46	+0.36	+0.72	+1.43	+0.60	+0.47	+0.32	+1.41	+1.16	
E119 { D	+1.39	+0.41	+0.41	+0.80	+1.43	+1.39	+0.67	+0.52	+0.77	+1.41	
E119 { C	+1.42	+0.47	+0.42	+0.72	+1.44	+1.35	+0.55	+0.41	+0.75	+1.43	
E119 { B	+0.45	+0.33	+0.27	+0.53	+0.61	+0.13	+0.14	+0.14	+0.13	+0.15	
E119 { A	+0.38	+0.35	+0.24	+0.58	+0.71	+0.12	+0.14	+0.13	+0.20	+0.13	

plate 2

60 min

	1	2	3	4	5	6	7	8	9	10	
	$\frac{1}{2}G_1$	$\frac{1}{2}G_{2a}$	$\frac{1}{2}G_{2b}$	$\frac{1}{2}G_3$	$\frac{1}{2}M$	$\frac{1}{2}G_1$	$\frac{1}{2}G_{2a}$	$\frac{1}{2}G_{2b}$	$\frac{1}{2}G_3$	$\frac{1}{2}M$	plate 2
E455 { F	+0.66	+0.56	+0.48	+0.85	+1.41	+0.88	+0.60	+0.44	+0.92	+1.47	} E102 F E D C B A
E455 { E	+0.56	+0.49	+0.43	+1.04	+1.41	+0.94	+0.54	+0.34	+0.80	+1.45	
E37 { D	+0.66	+0.64	+0.41	+0.79	+1.41	+0.68	+0.63	+0.41	+0.73	+1.43	
E37 { C	+0.61	+0.54	+0.37	+0.77	+1.44	+0.58	+0.53	+0.42	+0.79	+1.44	
E37 { B	+0.55	+0.38	+0.25	+0.55	+0.72	+0.12	+0.15	+0.12	+0.18	+0.20	
E37 { A	+0.51	+0.42	+0.24	+0.59	+0.69	+0.11	+0.17	+0.10	+0.33	+0.10	

Cytotoxicity to LNCaP

Cytotoxicity to L936P

Cytotoxicity to L9358P

[illegible]

cyto toxicity to Lmcrp

[illegible]

Cytotoxicity to LNCaP

[illegible]

# Exhibit H

Slide	Section	MoAb		Result	
					PT weak +
26	1 NK (unsoeld, W),	E27	E99	—	—
	2	E202	E455	—	—
	3	653	F31	—	+
35	4 Nor liver	E27	E99	—	—
	5 (31-53291)	E202	E455	—	—
	6	653	W6/32	—	+++
41	7 Nor lung (208)	E27	E99	—	—
	8	E202	E455	Alveola (-)	Bronchiole Basal (+)
	9	653	W6/32	—	+++
39	10 Nor pancreas (271)	E27	E99	+++	—
	11	E202	E455	—	—
	12	653	W6/32	—	+++
39	13 Nor Testis (259)	E27	E99	—	—
	14	E202	E455	—	—
	15	653	W6/32	—	connective tissue ++
52	16 Pca (WHEELER, A)	E27	E99	heterogen ++	weak +
	17	E202	E455	basal + - ++	basal ++
	18	653	P410	—	weak +
51	19 Pca (Culver)	E27	E99	heterogen + - ++	+ - ++
	20	E202	E455	heterogen basal +	heterogen basal +
	21	653	P410	—	+
51	22 BPH (Chan) Jam	E27	E99	—	+
	23	E202	E455	heterogen +	heterogen ++
	24	653	P410	—	++

[REDACTED]

Slide	Section	MoAb			
	1 Esophagus (31-57932)	E27	E99	—	—
6	2	E202	E455	basal +	basal +
	3	653	W6/32	—	+
	4 <sup>Nov</sup> Uterus (31-52931)	E27	E99	—	—
6	5	E202	E455	—	—
	6	653	W6/32	—	+
	7 Small bowel (31-51650)	E27	E99	++ - ++	—
	8	E202	E455	—	—
6	9	653	W6/32	—	+
	10 Stomach (31-53427)	E27	E99	—	—
8	11	E202	E455	section not good	
	12	653	W6/32	+	+
	13 Thyroid (P41-422)	E27	E99	—	—
		E202	E455	—	—
11	14	653	W6/32	—	+
	15				
	16 Spleen (P41-39)	E27	E99	++?	—
		E202	E455	—	—
11	17	653	W6/32	—	+
	18				
	19 Pca (Mahoney, w)	E27	E99	heterogen	+
		E202	E455	++	++
59	20	653	PA10	—	heterogen ++
	21				
	22 Pca Verclugo, J	E27	E99	—	++ - ++
		E202	E455	heterogen weak +	heterogen +
67	23	653	PA10	—	++ - ++
	24				

# Exhibit I

Slide Section		Mu Ab	Result		
1	NK, N.C., Lncap tumor	J-267	+	-	+
2		J-271	+	+	+
3		J-284	+	+	+
4		J-297	+	-	-
5		J-331	+	+	+
6		J-377	+	+	+
7		J-380	+	-	-
8		J-382	+	+	+
9		J-385	+	+	+
10		J-386	-	+	-
11		J-402	+	+	+
12		J-415	PT+	-	+
13		J-421	+	+	+
14		J-437	+	+	+
15		J-445	+	+	-
16		J-457	+	+	+
17		J-472	-	-	-
18		J-478	-	-	-
19		J-518	-	-	-
20		J-515	-	-	-
21		J-527	+	-	+
22		J-533	PT+	-	+
23		653	-	-	-
24		Eqg	PTT	-	+

# Exhibit J

SITE	Section	MSAL				
12	1 NK (Rico, Romo)	J-4	J-5	+		+
	2	J-14	J-96	+		+
	3	J-110	J-122	+		+
	4	J-254	J-262	-		-
	5	653	E31	-		+
33	6 NCR LIVER (31-53291)	J-4	J-5	+		+
	7	J-14	J-96	+		+
	8	J-110	J-122	+		+
	9	J-254	J-262	-		-
	10	653	E31	-		+
33	11 PCA (Lee, Tran)	J-4	J-5	+		+
	12	J-14	J-96	+		+
	13	J-110	J-122	+		+
	14	J-254	J-262	-		-
	15	653	E99	-		+
V	16 NK, N.C. LNCAP	J-791		-	-	+
	17	J-662		+	-	+
	18	J-722		-	-	-
	19	J-791		+	+	+
	20	J-797		-	+	+
	21	J-807		-	-	-
	22	J-812		+	-	-
	23	J-816		+	+	+
	24	J-823		+	+	+
	25	653		-	-	-
	26	E99		+	-	+



# Exhibit K

Slide	Section	MoAb		Result	
12	1 NK (R70, R70a)	J-415	J-533	PT+	PT+
	2 "	653	Eg9	—	PT+
35	3 Nor Liver (31-53291)	J-415	J-533	—	—
44	4 Nor Sm 2.5.5.10.10.10	J-415	J-533	—	—
	5 (LACRANCE E)	653	W6/2	—	+
41	6 Nor lung (20E)	J-415	J-533	—	—
	7 "	653	W6/2	—	+
59	8 BPH (Kramme: A)	J-415	J-533	1-3+	1-2+
	9 "	653	Eg9	—	1-3+
59	10 CPH (Jseug. Dand)	J-415	J-533	1-3+	1-2+
	11 "	653	Eg9	—	1-3+
65	12 PCA (Walsh, J)	J-415	J-533	2-4+	2-4+
	13 "	653	Eg9	—	2-4+
63	14 PCA (Lingau, E)	J-415	J-533	1-3+	1-3+
	15 "	653	Eg9	—	1-3+

# Exhibit L

Side	Section	M & A's		Result	
	1 Pca	E455	E99	0-2+	0-2+
	2 (CANNON, THOMAS)	J-415	J-533	0-3+	0-3+
	3	J-591	653	0-3+	-
23	4	H125	H647	-	0-3+
	5	H648	H108-3	0-3+	0-1+
	6	P410	P30	1+	3+
	7 Pca	E455	E99	-	3+
	8 (CHEN, VEN)	J-415	J-533	3+	3+
	9	J-591	653	3+	-
23	10	H125	H647	-	1-3+
	11	H648	H108-3	1-3+	2+
	12	P410	P30	2+	3+
	13 Pca	E455	E99	3+	0-2+
	14 (FLORES, FORTUNADO)	J-415	J-533	1-2+	1-2+
	15	J-591	653	1-2+	-
23	16	H125	H647	0-3+	0-2+
	17	H648	H108-3	0-2+	0-1+
	18	P410	P30	1-2+	3+
	19 Pca	E455	E99	0-1+	1-2+
	20 (LIAN YU CHANG)	J-415	J-533	1-2+	1-2+
	21	J-591	653	1-2+	-
27	22	H125	H647	Occasional 1+	3+
	23	H648	H108-3	3+	1+
	24	P410	P30	1-2+	3+
	25 Pca	E455	E99	4+	0-2+
	26 (O'Brien, William)	J-415	J-533	0-3+	0-3+
27	27	J-591	653	0-2+	-
27	28	H125	H647	0-3+	0-3+